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Poverty and the growth of emotional and conduct problems in children with autism with and without comorbid ADHD

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Abstract

We investigated the longitudinal relationship between socio-economic disadvantage (SED) and trajectories of emotional and conduct problems among children with autism spectrum disorder (ASD) who had comorbid attention deficit/hyperactivity disorder (ADHD; ASD+ADHD) or not (ASD-ADHD). The sample was 209 children with ASD who took part in the UK's Millennium Cohort Study. Trajectories of problems across ages 3, 5, and 7 years were analyzed using growth curve models. The ASD-ADHD group decreased in conduct problems over time but the ASD+ADHD group continued on a high trajectory. Although SED was not a risk factor for ASD+ADHD, it was associated with elevated emotional problems among children with ASD+ADHD. The effect of SED on emotional problems was not attenuated by parenting or peer problems.

The restriction that autistic spectrum disorder (ASD) and attention deficit/hyperactivity disorder (ADHD) cannot be dually diagnosed has been lifted in DSM-5 (American Psychiatric Association, 2013). However, estimates for prevalence rates of this comorbidity vary widely. Between 30% and 80% of children with a primary diagnosis of ASD are thought to display symptoms of ADHD (Rommelse, Franke, Geurts, Hartman, & Buitelaar, 2010; Simonoff et al., 2008), although more recent studies report rates around 20% (Musser et al. 2014; Russell, Rodgers, Ukoumunne, & Ford, 2014). What studies agree on is that children with comorbid ASD+ADHD symptoms are both more cognitively impaired (Karalunas, Geurts, Konrad, Bender, & Nigg, 2014; Russell et al., 2014; Tye et al., 2014; van der Meer et al., 2012; Yerys et al., 2009) and more likely to present with psychopathology, such as conduct problems/oppositionality and emotional problems (Jang et al., 2013).

In the ASD field, there is much research on the role of individual characteristics as risk factors for the development and maintenance of childhood psychopathology or general behaviour problems, but little on the role of risk factors in the broader context, including the family system. These few studies show that, in general, parenting stress (Bauminger, Solomon, & Rogers, 2010; Osborne, McHugh, Saunders, & Reed, 2008), controlling parenting (Boonen et al., 2014) and household chaos are positively related, whereas parental warmth (Midouhas, Yogaratnam, Flouri, & Charman, 2013) and limit setting (Osborne et al., 2008) are negatively related to problem behaviour in children with ASD. The findings about the role of family poverty in problem behaviour among children with ASD are more mixed. In a cross-sectional UK population-representative study, family material deprivation was not associated with psychiatric disorders (Simonoff et al., 2008) among 10-14 year olds with ASD, although it was associated with emotional problems at the age 16 follow-up (Simonoff et al., 2013). In another recent UK population study, family poverty was associated with emotional and behavioural problems in children with ASD at school entry, but its effect was

explained by lower parental warmth (Midouhas et al., 2013). Findings about the role of poverty or disadvantage in the persistence of emotional and behavioural problems in young people with ASD are also mixed. Whereas Taylor and Seltzer (2010) found that there was an association between higher income and greater improvement in emotional and behavioural well-being, others have found no effect (Emerson et al., 2014; Gray et al., 2012). To our knowledge, no study has investigated the role of family poverty and socio-economic disadvantage (SED) in general in the development and maintenance of problem behaviour in ASD children with and without comorbid ADHD. We carried out this study to fill this gap. We expected that poor children with comorbid ASD and ADHD would have more emotional and conduct problems than poor children with ASD only. The former group of children would have a greater accumulation of risk from both their comorbid status and stressors coming from their disadvantaged home environment, including poorer parenting and fewer economic and educational resources (Larson, Russ, Kahn, & Halfon, 2011).

Our study used a large longitudinal population sample of children with ASD in the UK, followed from preschool age to middle childhood. We were interested in exploring the role of SED in the development of problem behaviour in these two groups of children with ASD, but also in explaining how SED may be influencing this development. Being mindful of gaps in ASD research with regard to the role of the family but especially the peer group system in the maintenance and development of problem behaviour in children with ASD, we drew on research with typically developing children (Conger & Donnellan, 2007). Based on this evidence, we expected that SED would be linked to problem behaviour via poor relationships with peers (Due et al., 2009) and parents (Bradley & Corwyn, 2002). We expected these associations, even after controlling for maternal psychological distress, which is related to emotional (Simonoff et al., 2013) and behavioural (Totsika et al., 2013) problems in children with ASD. Maternal psychological distress is also strongly related to problem behaviour in

typically developing child populations, and fully or partially explains both the SED-problem behaviour link (Petterson & Albers, 2001) and the SED-harsh parenting association (Lovejoy, Graczyk, O'Hare, & Neuman, 2000). We also tested whether the relationship between SED and behaviour of children with ASD was confounded by the child's intellectual functioning. SED is strongly related to behaviour problems in children with an intellectual disability (Emerson et al., 2014; Emerson & Hatton, 2007). Intellectual disability is common in children with ASD. Approximately 50% of children with ASD have an intellectual disability (Charman et al., 2011).

Method

Sample

Data from Sweeps 2-4 (at ages 3, 5 and 7 years) of the Millennium Cohort Study (MCS) were used. MCS is a population-based cohort study of children born in the UK in 2000-2002, designed to over-represent families living in areas of high child poverty, areas with high proportions of ethnic minority populations across England, and the three smaller UK countries. Parent-reported data were collected through interviews and self-completion questionnaires. Ethical approval was gained from NHS Multi-Centre Ethics Committees, and parents gave informed consent before interviews took place. At Sweep 1 (when the children were aged 9 months), 18,522 families participated in MCS. The numbers of productive families at Sweeps 2, 3 and 4 were 15,590, 15,246 and 13,857, respectively. For families with twins and triplets, we used information only about the first-born twin or triplet. At Sweep 4, the main caregiver was asked, "Has a doctor or health professional ever told you that [Cohort child's name] had Autism, Asperger's Syndrome, or other autistic spectrum disorder?" The 'ASD' sample (n = 209, 174 boys) included children whose main caregiver indicated 'yes' to this question. The 'ASD+ADHD' sample (n = 44, 41 boys) were those children with ASD

whose main caregiver answered 'yes' to the question, also asked of all children at Sweep 4, "Has a doctor or health professional ever told you that [Cohort child's name] had Attention Deficit Hyperactivity Disorder (ADHD)?" The observed (weighted) prevalence was 1.7% for parent-reported ASD and 1.4% for parent-reported ADHD. A total of 19.9% of MCS children with ASD also had ADHD.

Measures

Emotional and conduct problems were our dependent (response) variables, and peer problems, harsh discipline, parent-child closeness and parent-child conflict our mediators. *Emotional, peer and conduct problems* were assessed at Sweeps 2-4 using the main caregiver's report of the Strengths and Difficulties Questionnaire (SDQ; Goodman, 1997), a valid and reliable measure of child psychopathology, and a useful clinical screening tool for both ASD and ADHD children (IIzuka et al., 2010; Russell, Rodgers, & Ford, 2013). Example items of the emotional problems scale include "Many worries, often seems worried" and "Nervous or clingy in new situations". For conduct problems, examples are "Often has temper tantrums or hot tempers" and "Often lies or cheats". As for peer problems, example items are "Rather solitary, tends to play alone" and "Picked on or bullied by other children". Total scores were calculated for each of these three 5-item (ranging from 0 to 2) scales. In our ASD sample, for each of these scales, Cronbach's alpha coefficients were sufficient in Sweep 4 (above .70) but low at Sweeps 2 and 3 (below .70), especially for peer problems. In particular, the coefficients for the emotional and conduct scales ranged from .57 to .76. The alphas for the peer problems scale were .65 and .70 at Sweeps 3 and 4, respectively, but only .48 at Sweep 2. Cut-off scores for the abnormal range - identifying 10% of the community population - are 4 for conduct problems, 5 for emotional symptoms, and 4 for peer problems (www.sdqinfo.org). At age 3, 55.5% of the ASD sample was in the abnormal range for conduct problems and 40.1% was in the abnormal range for peer problems. However, at 9.1%

the percentage of 3-year-old children in the abnormal range for emotional symptoms was aligned with community sample expectations.

Family *socio-economic disadvantage* (SED) was measured at Sweeps 2-4 with a 4-item summative index of family poverty (Malmberg & Flouri, 2011). The four dichotomous items were overcrowding (>1.5 people per room excluding bathroom and kitchen), lack of home ownership, receipt of income support, and income poverty (equivalised net family income being below the 60% of the national median household income). *Harsh discipline* was assessed using the main caregiver's response to Straus and Hamley's (1997) Conflict Tactics Scale at Sweep 2. This 7-item questionnaire asks how often the respondent uses various discipline techniques when the child misbehaves. The questionnaire covers a wide range of behaviours, namely ignoring, smacking, shouting, sending the child to his/her room, taking away treats, telling the child off, and bribing the child. Cronbach's alpha was .61. *Parent-child conflict and parent-child closeness* were assessed with the short form of the Child-Parent Relationship Scale (Pianta, 1992) at Sweep 2. The scale has 15 items rated on a 5-point Likert scale measuring conflict and closeness (α = .74 and .72, respectively). *Covariates* were gender, ethnicity (white or not), maternal education (University degree or not), and the two time-varying covariates of family structure (intact or not) and mother's psychological distress. Psychological distress was measured with the 6-item Kessler scale (Kessler et al., 2003), which assesses the experience of recent non-specific psychological distress (α = .82 to .90 across sweeps).

For our supplementary analysis, we measured the child's *intellectual functioning* with the age-adjusted scores of the cognitive ability measures administered to MCS children at Sweeps 2-4. At Sweep 2, these included the Bracken School Readiness Assessment-Revised (BSRA-R), which assesses children's 'readiness' for formal education by testing their knowledge and understanding of basic concepts (Bracken, 1998), and the British Ability

Scales (BAS; Elliot, Smith, & McCulloch, 1996) Naming Vocabulary scale which measures expressive language. At Sweep 3, ability was assessed with the BAS Naming Vocabulary, BAS Pattern Construction (measuring spatial problem solving) and BAS Picture Similarities (measuring non-verbal reasoning) scales. At age 7, MCS measured cognitive skills by maths achievement - assessed with the National Foundation for Educational Research (NFER) Progress in Maths - along with the BAS Pattern Construction and BAS Word Reading (measuring educational knowledge of reading) scales.

Analytic strategy

To predict children's emotional and conduct problems over time due to SED, we modelled individual trajectories of emotional and conduct problems using hierarchical linear regression models (also known, for longitudinal data, as growth curve models). Growth curve modelling allowed us to estimate mean trajectories (i.e., growth) of children's problems from age 3 to 7 by specifying an independent variable for time. In this study, our time variable was age in years centred at the grand mean across sweeps (5.28 years). Grand mean centring age minimises the correlation between age and age squared (which we had to include to capture the nonlinear pattern of scores for conduct problems, see below) with the effect of stabilising the estimation procedure (Raudenbush & Bryk, 2002). Importantly, as children with ASD may differ from each other in the level of problems at different ages, this approach also captures these individual differences in patterns over time and therefore deviations from mean trajectories. At the same time, it captures 'clustering' of repeated measures of children's psychopathology as an individual child's problems will be correlated across measurement occasions. In this type of model, occasions are considered to be at 'Level 1' and children at 'Level 2' (as occasions are nested within the child), and both fixed and random growth parameters are specified. The fixed parameters are the intercept (mean problem scores at the average age) and the slope (mean change in scores per annum). The random parameters

simultaneously capture the variation in scores between occasions for each child ('between-occasion variance') and between children at the average age ('between-child intercept variance') as well as the variation in their annual growth ('between-child slope variance'). The covariance between the intercept and the slope indicates whether there is a relationship between children's scores around age 5 and their growth between ages 3 and 7.

Data were analysed using SPSS, STATA and MLwiN. Missing data (the percentage of missing values per variable in our models ranged from 0 to 28%) were handled with multiple imputation, using the software REALCOM-Impute within MLwiN. This software was designed to implement multiple imputation for two-level continuous or categorical data. In descriptive analyses, survey weights were used to account for the MCS survey design and non-response. In multilevel analyses, the stratified sampling design of MCS was recognized by including the design variables that accounted for the oversampling of MCS children from particular area types. In the models, ADHD was introduced after SED to enable the investigation of the effect of SED on the emotional and conduct problems of children with ASD who had comorbid ADHD or not. The possible mediating role of harsh discipline, parent-child relationship and (time-varying) peer problems in the association between SED and conduct and emotional problems in both ASD groups was investigated after controlling for child and family covariates. The sequence of models fitted is shown in Table 1. Models 2-9 were conditional and therefore the variances and covariances of the growth curve components across children reflect residual variability, i.e., variability not accounted for by the considered covariates. In view of our small sample size (n = 209), we acknowledge that the results of the more complex models, such as those following Model 5, must be treated with caution.

For our supplementary analysis, we ran correlations between intellectual functioning and conduct and emotional problems. Then, as verbal ability was the only aspect of

intellectual functioning that was time-varying in our dataset (measured by BAS Naming Vocabulary at ages 3 and 5 and BAS Word Reading at age 7), we added it to Models 5-9 as a covariate (z-scored).

(Table 1)

Results

Descriptives

Table 2 describes our continuous and categorical variables in the full sample (n = 209), the ASD-ADHD sample (n = 165) and the ASD+ADHD sample (n = 44) at age 7. There were several differences between the two ASD groups on the study's variables (Table 2). Compared to children with ASD who did not have ADHD (ASD-ADHD), the ASD+ADHD comorbid group had higher conduct problem scores across all three sweeps and more emotional problems at age 7. At age 7, this group also had more distressed mothers. The comorbid group tended to be male and experience harsher discipline, was more likely to experience parental separation (but only at age 5), and was marginally more likely to have lower BAS Pattern Construction scores at age 5. There was no difference between the two ASD groups in either our summative index of SED or its individual components (Table 3).

(Tables 2 and 3)

We then calculated the mean conduct problem and emotional symptom scores at ages 3, 5 and 7 for children with ASD, with and without ADHD. In our sample of children with ASD, the average trajectory of emotional problems indicated an overall linear pattern with a steady increase in problems over time, whereas that of conduct problems was U-shaped. The trajectories of both problems appeared to differ by ADHD status, with children in the ASD+ADHD comorbid group showing higher levels of conduct problems and an acceleration

of emotional problems in older ages (Figures 1-2). For both types of problems, the trajectories were non-parallel, suggesting that emotional and conduct problems varied with age and between children. Therefore, a two-level growth curve model was deemed appropriate to capture inter-individual differences in intra-individual change. Because age was centred at the grand mean across the three sweeps, all main effects predicted emotional and conduct problems at the second measurement occasion (around age 5).

(Figures 1-2)

Growth Curve Regression Models

We ran growth curve models (Models 1-9) for emotional and conduct problems separately, and then examined whether the inclusion of child's verbal ability as a covariate altered the model results. Models 5 and 9 are presented in detail in Table 4. Results of Models 1-8 are presented in Supplementary Table 1.

Emotional problems. The unconditional model (with only age and age^2) showed significant variation in emotional problems between children with ASD at the average age and within children over time. In Model 2, there was a significant effect for SED at the average age but not on the trajectory. The variation in emotional problems between children around age 5 and between the three occasions remained significant. Looking at the random parameters, the between-child intercept-slope variance covariance showed that children with higher emotional problem scores around age 5 appeared to have a faster rate of growth in emotional problems over time compared to those children with lower emotional problem scores. Model 3 showed that ADHD had a nonsignificant main effect on emotional problems, but a significant effect on their annual change. Model 4 showed that the main effect of SED became nonsignificant and remained this way in all subsequent models. In this model, the positive effect of the interaction between SED and ADHD on emotional problems was

significant (suggesting that the effect of SED on emotional problems was stronger for the comorbid group), although the effect of this interaction on the annual change in emotional problems was not. The effect of the interaction between SED and ADHD was robust to adjustment for the covariates in Model 5 (Table 4). In this model, maternal psychological distress was also significantly associated with emotional problem scores, and remained significant in all subsequent models. Models 6-9 showed that neither harsh discipline nor peer problems or parent-child relationship quality explained the interaction between SED and ADHD on emotional problems. Model 9 (Table 4), which examined all these proposed mediators together, showed that the between-child intercept-slope variance covariance became nonsignificant. There were (positive) main effects for peer problems and parent-child closeness on emotional problems. Figure 3 plots this significant and robust SED x ADHD interaction effect on emotional problems. As can be seen, the comorbid group was at high risk for emotional problems but only if it was poor. Among non-poor families, the comorbid group was not at elevated risk for emotional problems.

(Table 4 and Figure 3)

Conduct problems. As explained, we captured the curvature of the average rate of change in conduct problems by including a fixed term for age squared in our models. The mean linear and quadratic fixed effects therefore indicate the average rate at which children change annually at around age 5 and the acceleration of that rate, respectively. Model 1 (the unconditional model) showed that there were significant variations in conduct problems between children with ASD and between the three occasions for each child. In Model 2, SED was associated with conduct problems at age 5 but not with their trajectory. Model 3 showed main effects for SED and ADHD on conduct problems. It also showed an effect for ADHD on the linear slope of conduct problems, which remained significant in Model 4. The other significant effect in Model 4 was that of the interaction between SED and ADHD. The

addition of the covariates in Model 5 (Table 4) rendered the SED x ADHD effect nonsignificant. Of the covariates entered, both maternal psychological distress and white ethnicity were related to conduct problems at the average age, and both remained significant in all subsequent models. Models 6 to 8 showed that harsh discipline, peer problems and parent-child conflict were related to conduct problems at age 5. Model 9 (Table 4 and Figure 4), which examined all proposed mediators together, showed that there was a main effect only for peer problems, although parent-child conflict was related to both the linear and the non-linear slope of conduct problems.

(Figure 4)

Supplementary analysis. We carried out some additional work to ensure that intellectual functioning was not confounding the association between SED and behaviour in children with ASD. In our sample, the association between cognitive abilities and emotional or conduct problems was weak. For example, none of the intellectual functioning variables available in MCS at age 3 years (i.e., Bracken School Readiness and BAS Naming Vocabulary) was significantly associated with age 3 conduct or emotional problems. At age 5, of the three intellectual functioning variables available (BAS Naming Vocabulary, BAS Pattern Construction and BAS Picture Similarities), only Pattern Construction was significantly related to behaviour (for both conduct and emotional problems, the association was -.22, $p < .01$). At age 7, all three intellectual functioning variables (NFER Progress in Maths, BAS Pattern Construction and BAS Word Reading) were related to emotional symptoms (rs were, respectively, -.19, -.18, and -.23), although none was related to conduct problems. Verbal ability, as a time-varying covariate in Model 5, was not related either to the intercept or the slopes of conduct problems. For emotional problems, verbal ability was related to both the intercept and the slope of problems. However, both effects became nonsignificant in Model 7, when peer problems were added, suggesting that peer problems

explained the association between low verbal ability and high emotional symptoms (results available on request).

Discussion

Although socio-economic disadvantage (SED) differences in ASD and comorbid ASD+ADHD have attracted attention (Rai et al., 2012; Russell et al., 2014), no study, to our knowledge, has examined the role of SED in the development of emotional and behavioural problems of children with ASD who had comorbid ADHD or not. Using data from the Millennium Cohort Study (MCS), a longitudinal general population study in the UK, we attempted to fill this gap. In line with previous research, we showed that children with comorbid ASD+ADHD symptoms have elevated conduct problems compared to children with ASD only (Holtmann et al., 2007; Jang et al., 2013). Importantly, we also found that these children were more likely to experience harsh parenting and maternal psychological distress than children with ASD who did not have ADHD (ASD-ADHD). However, the ASD+ADHD group did not differ from the ASD-ADHD group in SED, although children with ASD are more likely than children without ASD to come from socio-economically disadvantaged families (Midouhas et al., 2013).

Although SED was therefore not a risk factor for ASD+ADHD, it was associated, as expected, with significantly elevated (emotional) problems among children in this group. According to our findings, the ASD+ADHD comorbid group was at high risk for emotional problems but only if it was poor. Poverty may exacerbate such problems for children with both developmental disorders due to the accumulation of risk they experience. Among non-poor families, children with ASD+ADHD did not differ in emotional problems from children with ASD but without ADHD. Being from a non-poor background may act as a buffer to increases in problem behaviour in children with both conditions because poor families with

children with comorbid ASD+ADHD may not have sufficient resources to provide appropriate educational and social supports for them. This effect of SED on emotional problems appeared to be robust to adjustment for peer problems, harsh discipline and maternal psychological distress, but also parent-child relationship which has been shown to mediate the effect of SED on broad problem behaviour in children with ASD (Midouhas et al., 2013). By contrast, poverty did not modify the effect of ADHD status on conduct problems among children with ASD. While the ASD group without ADHD decreased in conduct problems over time, the comorbid group continued on a high trajectory. It would appear, therefore, that ADHD is a risk factor for a high conduct problems trajectory in children with ASD, and poverty is a risk factor for emotional symptoms in children with comorbid ASD+ADHD.

As well as building evidence around comorbidity between ASD, ADHD and psychiatric problems in children, our study produced some new findings about the emotional and behavioural development of children with ASD from early to middle childhood. For example, it showed that peer problems were associated with both emotional and conduct problems in children with ASD, and parent-child conflict was associated with a high conduct problems trajectory. Interestingly, parent-child closeness elevated the risk for (emotional) problems, perhaps in line with evidence that, in children with ASD, adjustment difficulties are linked both with parenting that is highly intrusive and lacking in warmth, and with emotional over-involvement (Greenberg, Seltzer, Hong, & Orsmond, 2006). Importantly, our study also supported previous findings about the role of maternal mental health difficulties in emotional and behavioural problems of children with ASD (Simonoff et al., 2013; Totsika et al., 2013).

Our study has some limitations we must acknowledge. First, as also reported by others who have used the MCS data (Russell et al., 2014), in MCS the prevalence of parent-reported

ASD in 7-year-olds in 2008 (1.7%) is high compared to around 1% in both US (Kogan et al., 2009) and other UK studies (Baird et al., 2006) around that time. Prevalence of (parent-reported diagnosis of) ADHD, by contrast, is low (1.4%) in MCS compared to almost 10% among 3-17 year-olds in the US (Bloom, Jones, & Freeman, 2013). In our study, 19.9% of the MCS 7-year-olds with ASD also had ADHD, in line with recent findings about rates in childhood ASD+ADHD co-diagnosis in non-UK population-based samples (Musser et al., 2014). Second, the severity of autism in our sample was unknown. Symptom severity can contribute significantly to the degree of psychiatric problems displayed by children with ASD (Tonge, Brereton, Gray, & Einfeld, 1999). Third, and related to this, we did not control for information about medication status. Medication status can affect both problem severity and parent reports, but has not yet been added to MCS. Fourth, the SDQ scales in the early years were less reliable than those at age 7, with the peer problems scale at age 3 being particularly problematic. Therefore, the findings about the effects of peer problems should be interpreted with caution. Fifth, both emotional and conduct problems and parent-child relationship and harsh discipline were parent-reported. Sixth, and most important, ASD and ADHD could not have been dually diagnosed in 2008. Parents may have inferred that a diagnosis was made if ASD or ADHD was suggested by a health professional but not confirmed by further assessment (Russell et al., 2014). Despite these limitations, our study's prospective longitudinal design allowed for a rigorous examination of the role of poverty and both family and peer relations as predictors of early trajectories of conduct and emotional problems in children with ASD who had comorbid ADHD or not.

Conclusion

Our study helps identify families with young children with ASD to whom clinical services should offer support and intervention. Our study investigated the trajectories of

emotional and conduct problems of young children with ASD who had comorbid ADHD or not, and explored the role of family poverty in these trajectories. Compared to children with ASD who did not have ADHD, children with comorbid ASD+ADHD appear to be on a high conduct problems trajectory. Peer problems and maternal psychological distress were strong predictors of both emotional and conduct problems in children with ASD. Poverty was a risk factor for emotional symptoms in the comorbid ASD+ADHD group: children with comorbid ASD+ADHD were a vulnerable group for emotional symptoms but only if they were poor.

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Table 1

Model Summary (Emotional and Conduct Problems)

Models	Specification
Model 1 (unconditional)	Age (grand mean centred) in years[a]
Model 2	Model 1 + design variables[b] + socio-economic disadvantage (SED)+ SED x age
Model 3	Model 2 + ADHD + ADHD x age
Model 4	Model 3 + SED x ADHD + SED x ADHD x age
Model 5	Model 4 + child[c] and family[d] covariates
Model 6	Model 5 + harsh discipline + harsh discipline x age
Model 7	Model 5 + peer problems + peer problems x age
Model 8	Model 5 + parent-child conflict + parent-child conflict x age + parent-child closeness + parent-child closeness x age
Model 9	Model 8 + harsh discipline + harsh discipline x age + peer problems + peer problems x age

[a]In the models for conduct problems, age-squared was also added to account for the U-shape of the average trajectory of conduct problems. Therefore, for conduct problems, growth was modelled by interactions with both age and age-squared. [b]This is the 'stratum' variable which accounts for the MCS sampling design. The nine MCS strata are 'England-advantaged', 'England-disadvantaged', England-ethnic', 'Wales-advantaged', 'Wales-disadvantaged', 'Scotland-advantaged', 'Scotland-disadvantaged', 'Northern Ireland-advantaged', and 'Northern Ireland-disadvantaged'. [c]Gender and ethnicity (white or not). [d]Maternal education (University degree or not), time-varying family structure (intact or not) and time-varying maternal psychological distress.